What is claimed is:

1. A method of modulating binding interaction between a first molecule which is not LFA-1 or an I domain-containing fragment thereof, and a binding partner molecule, said first molecule comprising an α/β domain structure, said α/β structure comprising an allosteric regulatory site, said method comprising the step of contacting said first molecule with an allosteric effector molecule that interacts with said allosteric regulatory site and promotes a conformation in a ligand binding domain of said α/β structure that modulates binding between said first molecule and said binding partner molecule.

2. A method of modulating binding interaction between a first molecule which is not LFA-1 or an I domain-containing fragment thereof, and a binding partner molecule, said first molecule comprising an α/β domain structure, said α/β structure comprising an allosteric regulatory site, said method comprising the step of contacting said first molecule with an allosteric effector molecule, said allosteric effector molecule comprising a diaryl compound, said diaryl compound interacting with said allosteric regulatory site and promoting a conformation in a ligand binding domain of said α/β structure that modulates binding between said first molecule and said binding partner molecule.

3. A method of modulating binding interaction between a first molecule which is not LFA-1 or an I domain-containing fragment thereof, and a binding partner molecule, said first molecule comprising an α/β domain structure, said α/β structure comprising an allosteric regulatory site, said method comprising the step of contacting said first molecule with an allosteric effector molecule, said allosteric effector molecule selected from the group consisting of diaryl sulfide compounds and diarylamide compounds, said allosteric effector molecule interacting with said allosteric regulatory site and promoting a conformation in a ligand binding domain of said α/β structure that modulates binding between said first molecule and said binding partner molecule.

4. The method of claim 1, 2, or 3 wherein said first molecule comprises a Rossmann fold structure, said Rossmann fold structure comprising said allosteric regulatory site.

5. The method of claim 4 wherein said Rossmann fold structure in said first molecule comprises a β sheet having β sheet strands positioned in a 321456 or 231456 orientation.

6. The method of claim 4 wherein said Rossmann fold structure in said first molecule comprises a β sheet having β sheet strands positioned in a 3214567 orientation.

7. The method of claim 4 wherein said Rossmann fold structure in said first molecule comprises a β sheet having β sheet strands positioned in a 32145 orientation.

8. The method of claim 1, 2, or 3 wherein said first molecule comprises an I domain structure.

9. The method of claim 1, 2, or 3 wherein said first molecule comprises an A domain structure.

10. A method of modulating binding interaction between a first molecule and a binding partner molecule, said first molecule having an amino acid sequence which exhibits less than about 90% identity to the LFA-1 I domain amino acid sequence set out in FIGURE 1, said first molecule comprising an α/β structure, said α/β domain structure comprising an allosteric regulatory site, said method comprising the step of contacting said first molecule with an allosteric effector molecule that interacts with said allosteric regulatory site and promotes a conformation in a ligand binding domain of said α/β structure that modulates binding between said first molecule and said binding partner molecule.

11. A method of modulating binding interaction between a first molecule and a binding partner molecule, said first molecule having an amino acid sequence which exhibits less than about 90% identity to the LFA-1 I domain amino acid sequence set out in FIGURE 1, said first molecule comprising an α/β structure, said α/β domain structure comprising an allosteric regulatory site, said method comprising the step of contacting said first molecule with an allosteric effector molecule, said allosteric effector molecule comprising a diaryl compound, said diaryl compound interacting with said allosteric regulatory site and promoting a conformation in a ligand binding domain of said α/β structure that modulates binding between said first molecule and said binding partner molecule.

12. A method of modulating binding interaction between a first molecule and a binding partner molecule, said first molecule having an amino acid sequence which exhibits less than about 90% identity to the LFA-1 I domain amino acid sequence set out in FIGURE 1, said first molecule comprising an α/β domain structure, said α/β structure comprising an allosteric regulatory site, said method comprising the step of contacting said first molecule with an allosteric effector molecule, said allosteric effector molecule selected from the group consisting of diaryl sulfide compounds and diarylamide compounds, said allosteric effector molecule interacting with said allosteric regulatory site and promoting a conformation in a ligand binding domain of said α/β structure that modulates binding between said first molecule and said binding partner molecule.

13. The method of claim 10, 11, or 12 wherein said first molecule has an amino acid sequence that exhibits a percent identity with respect to the LFA-1 I domain amino acid sequence less than about 40%, about 45%, about 50%, about 55%, about 60%, about 65%, about 70%, about 75%, about 80%, about 85%, or about 90%.

14. The method of claim 10, 11, or 12 wherein said first molecule comprises a Rossmann fold structure, said Rossmann fold structure comprising an allosteric regulatory site.

15. The method of claim 14 wherein said first molecule has an amino acid sequence that exhibits a percent identity with respect to the LFA-1 I domain amino acid sequence less than about 40%, about 45%, about 50%, about 55%, about 60%, about 65%, about 70%, about 75%, about 80%, about 85%, or about 90%.

16. The method of claim 14 wherein said Rossmann fold structure in said first molecule comprises a β sheet having β sheet strands positioned in a 321456 or 231456 orientation.

17. The method of claim 16 wherein said first molecule has an amino acid sequence that exhibits a percent identity with respect to the LFA-1 I domain amino acid sequence less than about 40%, about 45%, about 50%, about 55%, about 60%, about 65%, about 70%, about 75%, about 80%, about 85%, or about 90%.

18. The method of claim 14 wherein said Rossmann fold structure in said first molecule comprises a β sheet having β sheet strands positioned in a 3214567 orientation.

19. The method of claim 18 wherein said first molecule has an amino acid sequence that exhibits a percent identity with respect to the LFA-1 I domain amino acid sequence less than about 40%, about 45%, about 50%, about 55%, about 60%, about 65%, about 70%, about 75%, about 80%, about 85%, or about 90%.

20. The method of claim 14 wherein said Rossmann fold structure in said first molecule comprises a β sheet having β sheets strands positioned in a 32145 orientation.

21. The method of claim 20 wherein said first molecule has an amino acid sequence that exhibits a percent identity with respect to the LFA-1 I domain amino acid sequence less than about 40%, about 45%, about 50%, about 55%, about 60%, about 65%, about 70%, about 75%, about 80%, 85%, or about 90%.

22. The method of claim 10, 11, or 12 wherein said first molecule comprises an I domain structure.

23. The method of claim 23 wherein said first molecule has an amino acid sequence that exhibits a percent identity with respect to the LFA-1 I domain amino acid sequence less than about 40%, about 45%, about 50%, about 55%, about 60%, about 65%, about 70%, about 75%, about 80%, about 85%,or about 90%.

24. The method of claim 10, 11, or 12 wherein said first molecule comprises an A domain structure.

25. The method of claim 24 wherein said first molecule has an amino acid sequence that exhibits a percent identity with respect to the LFA-1 I domain amino acid sequence less than about 40%, about 45%, about 50%, about 55%, about 60%, about 65%, about 70%, about 75%, about 80%, about 85%, or about 90%.

26. The method of any one of claims 1-3, 5-7, 10-12, 15-21, 23 or 25 wherein the modulator promotes a conformation in the ligand binding domain of said

first molecule that increases binding between said first molecule and said binding partner molecule.

27. The method of claim 26 wherein the increase in binding between the first molecule and the second molecule results in increased enzymatic activity of the first molecule.

28. The method of any one of claims 1-3, 5-7, 10-12, 15-21, 23 or 25 wherein the modulator promotes a conformation in the ligand binding domain of said first molecule that decreases binding between said first molecule and said binding partner molecule.

29. The method of claim 28 wherein the decrease in binding between the first molecule and the second molecule results in decreased enzymatic activity of the first molecule.

30. The method of any one of claims 1-3, 5-7, 10-12, 15-21, 23 or 25 wherein the first molecule is selected from the group consisting of the proteins set forth in Table 1.

31. The method of claim 30 wherein the first molecule is a eukaryotic molecule.

32. The method of claim 30 wherein the first molecule is a human molecule.

33. The method of claim 30 wherein the first molecule is a prokaryotic molecule.

34. The method of claim 30 wherein the first molecule is a bacterial molecule.

35. The method of claim 30 wherein the first molecule is selected from the group consisting of $\alpha_M\beta_2$, complement protein C2, complement protein Factor B, $\alpha_E\beta_7$, $\alpha_4\beta_7$, $\alpha_V\beta_3$, $\alpha_4\beta_1$, $\alpha_d\beta_2$,von Willebrand factor, Rac-1, HPPK, ftsZ, and ENR.

36. The method of claim 35 wherein the first molecule is $\alpha_M\beta_2$ and the binding partner protein is fibrinogen.

37. The method of claim 35 wherein the first molecule is $\alpha_M\beta_2$ and the binding partner protein is iC3b.

38. The method of claim 35 wherein the first molecule is $\alpha_E\beta_7$ and the binding partner protein is E-cadherin.

39. The method of claim 35 wherein the first molecule is $\alpha_4\beta_7$ and the binding partner protein is MAdCAM-1.

40. The method of claim 35 wherein the first molecule is $\alpha_V\beta_3$ and the binding partner protein is vitronectin.

41. The method of claim 35 wherein the first molecule is $\alpha_4\beta_1$ and the binding partner protein is VCAM.

42. The method of claim 35 wherein the first molecule is $\alpha_d\beta_2$ and the binding partner protein is VCAM.

43. The method of claim 35 wherein the first molecule is von Willebrand factor and the binding partner protein is gpIb.

44. The method of claim 35 wherein the first molecule is complement protein C2 and the binding partner protein is complement protein C4b.

45. The method of claim 35 wherein the first molecule is complement protein Factor B and the binding partner protein is complement protein C3b.

46. The method of claim 35 wherein the first molecule is Rac-1 and the binding partner is GTP.

47. The method of claim 35 wherein the first molecule is HPPK and the binding partner is ATP or HMDP.

48. The method of claim 35 wherein the first molecule is ftsZ and the binding partner is GTP.

49. The method of claim 35 wherein the first molecule is ENR and the binding partner is NADH.